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Dean T. Mason, MD, Editor-in-Chief

- **CLINICAL INVESTIGATIONS, 495**
  - Effects of coronary thrombolysis on signal-averaged ECG post AMI
  - ECG and enzymatic indicators of therapeutic success post IV streptokinase in AMI
  - Role of platelet activating factor in pathogenesis of myocardial reperfusion injury
  - Significance of collateral circulation in reversible LV asynergy by nitroglycerin with recent AMI
  - Clinical and angiographic restenosis post PTCA for nonacute total occlusions: Comparison of 1- vs 5-minute inflations
  - High-dose contrast agent administration during complex PTCA
  - Cardiac phase-related variability of densitometric quantification of post-PTCA lumens
  - Comparative sensitivity of intracoronary acetylcholine-induced spasm in anginal syndromes
  - Exercise-induced myocardial ischemia with vs without pathologic Q waves in CAD
  - Significance of exercise-induced ST changes in previous inferior myocardial infarction
  - Percutaneous mitral valvuloplasty with single rubber-nylon balloon: Long-term results in 71 patients
  - Percutaneous balloon valvuloplasty for mitral stenosis vs restenosis
  - Adrenergic hypersensitivity post abrupt withdrawal of propranolol vs nadolol: Influence of half-life differences
  - Influence of β-blockade on exercise hemodynamic response by Doppler-echo
  - Cardiopulmonary exercise testing post conditioning in older coronary patients
  - Myocardial calcium-dependent fluorescence transients during VF
  - Quantification of concealed AV node conduction in atrial fibrillation
  - Response of ouabain-induced arrhythmias to pacing: Acceleration instead of termination
  - Immunohistochemical characterization of ANP in ventricles with myocarditis or AMI of Kawasaki disease
  - Systemic and regional hemodynamic effects of α-blockade in chronic LV dysfunction
  - Sustained benefit of 72-hour nitroglycerin infusion in severe CHF
  - Doppler-echo quantification of aortic and mitral regurgitation
  - Clinical evaluation of prospective protocol for surgical timing in chronic aortic regurgitation
  - TEE identification of aortic arch protruding atherosclerotic plaque in systemic embolization
- **CURRICULUM AND PROGRESS IN CARDIOLOGY, 661**
  - Idiopathic ventricular fibrillation
  - Diabetes and fibrinogen related to cardiovascular risk: Framingham Study
  - Percutaneous fiberoptic LV angioscopy in myocarditis vs dilated cardiomyopathy
- **BRIEF COMMUNICATIONS, 688**
- **SUPPLEMENT, 719**
  - New insights into the management of myocardial ischemia

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Protruding atherosclerotic plaque in the aortic arch of patients with systemic embolization: A new finding seen by transesophageal echocardiography

Patients with unexplained stroke or other embolic phenomena are often referred for echocardiography. The aortic arch is not usually visualized in detail during routine echocardiography; however, with the introduction of transesophageal echocardiography, this area may be seen with great resolution. We recently studied three patients who had embolic events, and transesophageal echocardiography showed a new and unexpected finding; large, protrusive plaques in the aortic arch and descending aorta, which have mobile projections that move freely with the blood flow. These lesions could be responsible for embolic syndromes, especially after catheter manipulation in the aorta. (Am Heart J 1990;120:658.)

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Unexplained stroke is a common reason for referral of patients to the echocardiography laboratory for the evaluation of a possible cardiac source of embolization. Unfortunately routine echocardiography is frequently unyielding in such patients.[1] We have recently studied three patients with neurologic and peripheral arterial syndromes in whom transesophageal echocardiographic findings included striking and unexpectedly large protruding atherosclerotic plaques in the aortic arch and descending thoracic aorta with mobile components that could possibly be responsible for the clinical syndromes.

METHODS AND RESULTS

Patient 1. A 68-year-old woman with atrial fibrillation and bilateral carotid bruits had an episode of transient dysarthria and an embolus to one toe. Results of a computerized tomographic scan of the head were negative, and transthoracic echocardiographic findings showed no cardiac source for embolization. Results of transesophageal echocardiography showed a normal aortic valve and a normal-appearing mitral valve with mild-to-moderate mitral regurgitation. The left atrial appendage was free of thrombi and there was no atrial septal defect. In the descending thoracic aorta and aortic arch there were protruding, large atherosclerotic plaques, with projections that were flail and moving freely in the lumen with the blood flow (Fig. 1, *A*).

Patient 2. A 77-year-old woman had increasing angina pectoris. Results of cardiac catheterization showed severe circumflex coronary obstruction, moderate right coronary obstruction, and mild disease in the left anterior descending coronary artery. At the end of the procedure she had a cerebellar infarction. Results of non-invasive carotid artery studies and transthoracic echocardiography were unremarkable, and left ventricular angiographic findings showed no thrombus. Because of the angina, a repeat catheterization was considered for coronary angioplasty, but a transesophageal echocardiography was done first to evaluate the aorta. Results of this study showed large protruding atherosclerotic plaques in the aortic arch with a small mobile projection from one plaque (Fig. 1, *B*). Because of this and the prior cerebellar infarction, angioplasty was not done and the patient has been stable on a medical regimen for the angina.

Patient 3. A 70-year-old man was referred for transesophageal echocardiography to look for a cardiac source of embolization after he had had a transient neurologic event with staggering, diplopia, and a visual field defect. Results of routine transthoracic echocardiography were normal except for mild mitral regurgitation. Transesophageal echocardiographic findings showed a normal heart except for the mitral regurgitation, with no clots in the left atrial appendage and no atrial septal defect. In the aortic arch and descending aorta there were large atherosclerotic plaques with freely mobile projections similar to those seen in the two preceding patients (Fig. 1, *C*). Results of noninvasive carotid artery studies showed total occlusion of the right carotid and 90% stenosis of the left carotid artery.

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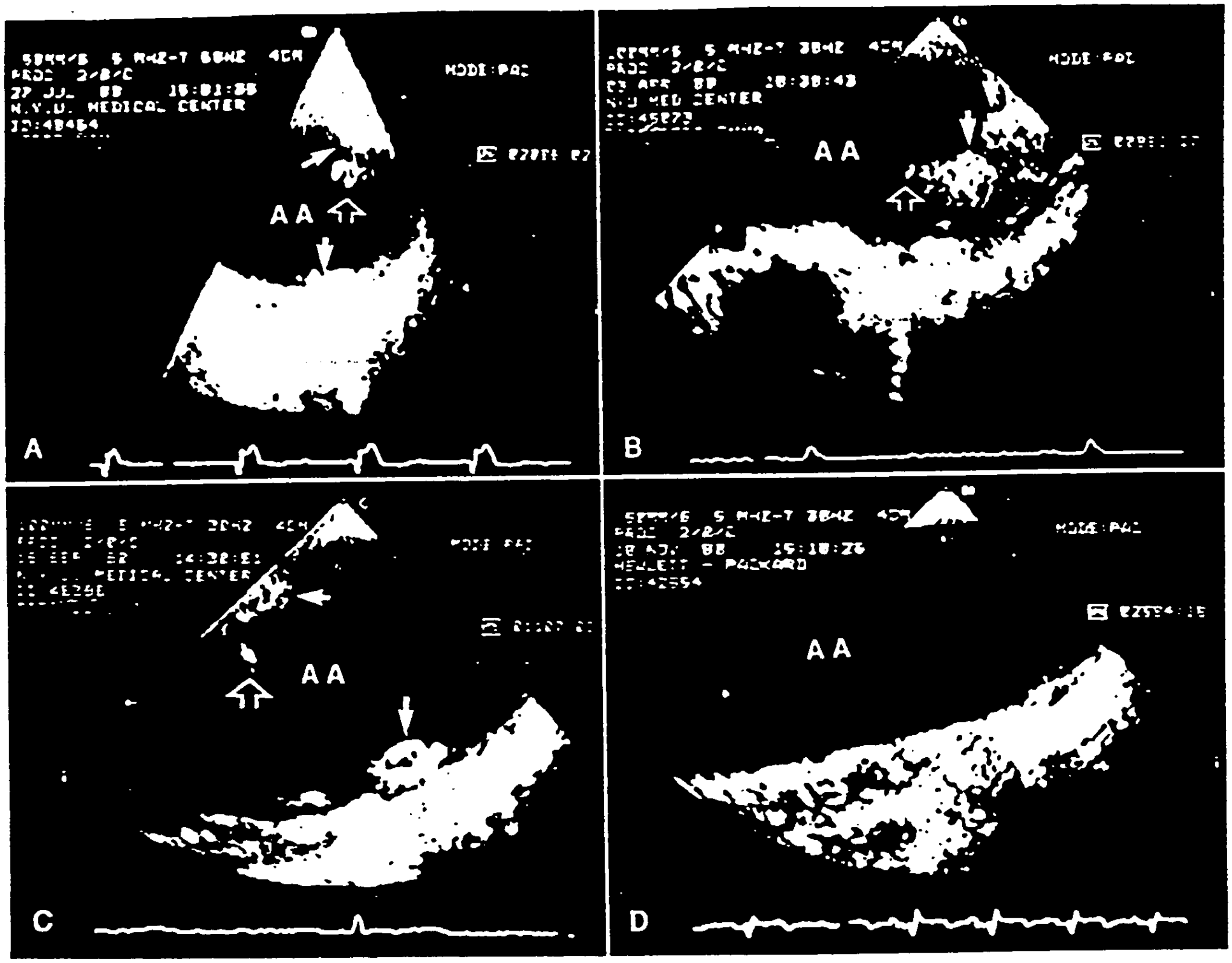

Fig. 1. Transesophageal echocardiogram showing aortic arch. *A*, Patient 1; *B*, Patient 2; *C*, Patient 3; *D*, normal patient (not in study) shown for comparison. *AA*, Aortic arch; *solid arrows*, projecting atherosclerotic plaque; *open arrows*, mobile projections from plaques.

DISCUSSION

Atheroemboli have been recognized as a cause of arterial obstruction either of the microvasculature or involving larger aortic branches, and they are especially common after surgical or catheter manipulation of an atherosclerotic abdominal aorta[2] and after catheterization of the left side of the heart.[3] This entity is an unusual cause of renal failure[4] and may also involve the retinal arteries,[5, 6] gastrointestinal tract,[7] and lower extremities, causing the blue toe sydrome.[8] It may also rarely result from blunt abdominal trauma.[9] Atheroembolization is often lethal, and the mortality rate was reported as 81% in one large review.[10]

Carotid artery atherosclerosis is commonly associated with cerebral infarction; however, the location of the bruit does not necessarily predict the location of the cerebral event, as pointed out in the Framingham study.[11] Furthermore, there is a group of patients without obvious carotid artery or cardiac disease who have unexplained strokes, and although results of routine echocardiography may show thrombus, vegetations, myxoma, or patent foramen ovale,[12] findings are frequently negative in such patients.[1] In patients in whom transthoracic echocardiographic findings are negative, transesophageal echocardiography may be useful because of its ability to image the heart in more detail. Paradoxical (right-to-left) shunting has been demonstrated across an atrial septal defect during the Valsalva maneuver by transesophageal echocardiography,[13] and thrombi in the left atrial appendage have also been seen via transesophageal echocardiography in patients with unexplained arterial embolization.[14]

Because of this, patients are often referred to the echocardiography laboratory for transesophageal echocardiography who have had unexplained neurologic events, and in the three patients discussed here the presence of strikingly large and protruding plaques with mobile portions in the thoracic aorta presents a possible cause of their clinical syndromes. These three patients represent 1% of the first 300 patients who have had transesophageal echocardiography in our laboratory, approximately one third of whom were referred for evaluation for a possible cardiac source of emboli. Patient 1 had had emboli to both the central nervous system and the lower leg, and she had bilateral carotid bruits. Patient 2 had a cerebellar infarction related to catheter manipulation in the aorta and had no evidence of carotid artery disease. Patient 3 had evidence of an event related to the posterior cerebral circulation and also had severe bilateral occlusive carotid disease. Thus transesophageal echocardiography has added another possible cause for these and similar embolic events and further points out the fact that demonstrable carotid disease may not be the only cause of the clinical events observed in patients with transient ischemic events or strokes.

Inasmuch as we do not have pathologic correlations, the composition of the mobile portions seen in these three patients is undetermined. However, it is likely that these findings represent the same process described in the pathologic literature as complex atheromas, which may include superimposed thrombus.[15] Thrombus has also been visualized by ultrasound in atherosclerotic abdominal aortic aneurysms.[16] In addition it is possible that the mobile portions seen represent pieces of the atheroma itself rather than clot. In an autopsy series of 267 patients, cholesterol embolization was found in 12.3% of those with severely eroded aortic plaques but in only 1.3% of those with moderately eroded plaques and in none of those with uneroded plaques.[17] In either case it is easy to see how this material could potentially be the source for embolization, especially during catheter manipulation in the aorta.

In conclusion, we have described a new transesophageal echocardiographic finding in patients with neurologic events and systemic arterial embolization. Treatment of such patients is problematic, as it has been suggested that anticoagulation may actually worsen the clinical syndrome, possibly by causing further plaque hemorrhage and thrombus superimposition.[18] In fact atheroembolization may even result from thrombolytic therapy.[19] It is interesting to speculate that such disease in the aortic arch might be responsible for some of the embolic events in the group of patients who do have carotid artery disease, as well as in the group with totally unexplained events. Transesophageal echocardiography has recently been reported to have a higher yield than routine transthoracic echocardiography in patients with systemic emboli (potential sources of embolization were detected in 65% of transesophageal studies but in only 37% of transthoracic studies in 479 patients).[20] Our three patients show a new and unusual finding, which may be especially important in patients in whom catheter manipulation in the aorta is contemplated.

REFERENCES

1. Greenland P, Knopman DS, Mikel FL, Asinger RW, Anderson DC, Good DC. Echocardiography in diagnostic assessment of stroke. Ann Intern Med 1981;95:51-3.
2. Braunwald E ed. Heart disease: A textbook of cardiovascular medicine. Philadelphia: WB Saunders Company, Harcourt Brace Jovanovich, Inc, 1988:1571, 1835.
3. Ramirez G, O'Neill WM, Lambert R, Bloomer HA. Cholesterol embolization: a complication of angiography. Arch Intern Med 1978;138:1430-2.
4. Smith MC, Ghose MK, Henry AR. The clinical spectrum of renal cholesterol embolization. Am J Med 1981;71:174-80.
5. Coppetto JR, Lessell S, Greco TP, Eisenberg MS. Diffuse disseminated atheroembolism. Arch Ophthalmol 1984;102:255-8.
6. Hollenhorst RW. Vascular status of patients who have cholesterol emboli in the retina. Am J Ophthalmal 1966;61: 1159-65.
7. Harvey RL, Doberneck RC, Black WC. Infarction of the stomach following atheromatous embolization. Report of a case and literature review. Gastroentrology 1972;62:469-72.
8. Fisher DF, Clagett GP, Brigham RA, Orecchia PM, Yonkey JR, Aronoff RJ, Fry RE, Fry WJ. Dilemmas in dealing with the blue toe syndrome: aortic vs. peripheral source. Am J Surg 1984;148:836-9.
9. Hertzer NR. Peripheral atheromatous embolization following blunt abdominal trauma. Surgery 1977;82:244-7.
10. Fine MJ, Kapoor W, Falanga V. Cholesterol embolization syndrome: a review of 221 cases in the English literature. Angiology 1987;38:769-84.
11. Wolf PA, Kannel WB, Sorlie P, McNamara P. Asymptomatic carotid bruit and risk of stroke. The Framingham study. JAMA 1981;245:1442-5.
12. Lechat P, Mas JL, Lascault G, Loron P, Theard M, Klimczak M, Drobinski G, Thomas D, Grosgogeat Y. Prevalence of patent foramen ovale in patients with stoke. N Engl J Med 1988;318:1148-52.
13. Tunick PA, Daniel W, Goldfarb A, Kronzon I. Paradoxical (right-to-left) shunting across an iatrogenic atrial septal defect during Valsalva's maneuver: a transesophageal Doppler echocardiographic study. J Am Soc Echo 1988;1:455-7.
14. Mugge A, Daniel WG, Wenzlaff P, Lichtlen PR. Patent foramen ovale or left atrial thrombi in unexplained arterial embolism. Lancet 1989;1:282-3.
15. Anderson JR ed. Muir's textbook of pathology, 12th ed. Baltimore: Edward Arnold, 1985:14.5, 14.6, 14.8.
16. Leopold GR, Goldberger LE, Bernstein E. Ultrasonic detection and evaluation of abdominal aortic aneurysms. Surgery 1972;72:939-45.
17. Flory CM. Arterial occlusions produced by emboli from eroded aortic atheromatous plaques. Am J Pathol 1945;21:549-65.
18. Moldveen-Geronimus M, Merriam Jr JC. Cholesterol embolization: from pathologic curiosity to clinical entity. Circulation 1967;35:946-53.
19. Ridker PM, Michel T. Streptokinase therapy and cholesterol embolication. Am J Med 1989;87:357-8.
20. Daniel WG, Angerman C, Engberding R, Erbel R, Hanrath P, Iliceto S, Kasper W, Visser C. Transesophageal echocardiography in patients with cerebral ischemic events and arterial embolism—a European multicenter study [Abstract]. Circulation 1989;80:II-473.